Treatment for hyperkalemia in hyporeninemic hypoaldosteronism

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To the Editor: A recently published report by Takemoto *et al.*[1] showed nice images of amyloid deposition in the juxtaglomerular apparatus. Although the authors attributed hyporeninemia to the amyloid deposition in the reported case, other conditions of the case might have contributed to hyporeninemia; hyporeninemic hypoaldosteronism can be seen in several conditions, including advanced age, without the amyloid deposition.[2] In these conditions, the blood volume is usually expanded rather than depleted, and fludrocortisone should not be used.

Hypoaldosteronism induces salt-wasting in renal distal tubules, which can cause hyperkalemia, but with normal renal function it typically will not induce hyperkalemia. Hyperkalemia is commonly seen in association with renal insufficiency, which is frequently accompanied by the volume expansion. Therefore, the reported patient, who had hyperkalemia with an estimated glomerular filtration ratio of $\sim$40 ml/min per 1.73 m^2, is more likely to have had the expanded volume at the time of diagnosis. With the volume expansion, medication that facilitates the distal tubular delivery of chloride, such as loop diuretics, would be appropriate, and medication that facilitates the distal tubular sodium reabsorption, such as fludrocortisone, could have exacerbated the volume expansion.

In addition, during treatment by fludrocortisone, serum sodium concentration should be monitored. Hyponatremia is a feature of renal salt-wasting; the volume depletion stimulates anti-diuretic hormone secretion. In severe hyponatremia, increasing serum sodium concentration faster than 8 mmol/l per day, should be avoided to reduce the risk of central pontine myelinolysis.[3]

1. Takemoto F, Ubara Y, Kaname S *et al.* Hyporeninemic hypoaldosteronism from secondary amyloidosis. *Kidney Int* 2008; **74**: 542.
2. Rosenberg ME, Smith LJ, Correa-Rotter R *et al.* The paradox of the renin-angiotensin system in chronic renal disease. *Kidney Int* 1994; **45**: 403–410.
3. Reynolds RM, Padfield PL, Seckl JR. Disorders of sodium balance. *BMJ* 2006; **332**: 702–705.

Masayuki Tanemoto[1]

[1]*Division of Nephrology, Hypertension, and Endocrinology, Department of Medicine, Tohoku University Graduate School of Medicine, Miyagi, Japan*

Correspondence: *Masayuki Tanemoto, Division of Nephrology, Hypertension, and Endocrinology, Department of Medicine, Tohoku University Graduate School of Medicine, 1–1 Seiryo-cho, Aoba-ku, Sendai, Miyagi 980-8574, Japan.*
E-mail: mtanemoto-tky@umin.ac.jp

Response to 'Treatment for hyperkalemia in hyporeninemic hypoaldosteronism'

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We read the comment by Tanemoto. with profound interest. The writer states that hyporeninemic hypoaldosteronism may be because of volume expansion in our case, and that fludrocortisones should not be used in such a condition. Regarding the issue of volume status, extra-cellular volume in our patient was not expanded because edema, body weight gain (changes in body weight were being monitored in outpatient visits), and cardiomegaly in chest X-ray were not found. Moreover, neither the jugular vein nor the vena cava were found to be dilated in abdominal echography. These findings together indicated the volume status in our patient was not expanded in any respect. Rather, she showed volume depletion, as evidenced by her serum uric acid level of 7.1 mg/dl. We understand that this is a kind of chicken and egg problem, but we are sure that she was volume-depleted without any significant proof of volume expansion.

The pathophysiology of the hyporeninemic hypoaldosteronism in our patient may need more rigorous investigation, but we attributed it to the direct inhibition of renin production in juxtaglomerular apparatus by massive amyloid deposition, as can be seen in Figure 1 of our article.[1] Stimulation test of renin secretion by upright posture and furosemide might have been better performed. Anyhow, the cause of an undetectable renin content in our case was ascribed to the problem of the juxtaglomerular apparatus similar to the condition, which is associated frequently with diabetes mellitus. Hyporeninemic hypoaldosteronism is seen mostly in diabetes mellitus.[2]

As to how hyporeninemic hypoaldosteronism is treated in clinical medicine, several measures are postulated,[2,3,] and fludrocortisones seemed the reasonable drug of choice in our case as her volume status was not depleted. Diuretics were not administered, as she was not edematous or showed deterioration of renal function probably because of intravascular volume depletion. In fact, fludrocortisone has been shown effective in patients with hyporeninemic hypoaldosteronism.[2] We successfully used fludrocortisone to correct life-threatening hyperkalemia without any adverse effect. The result also supported the idea that she was not volume-expanded, otherwise the drug might have induced hypervolemic complications such as peripheral edema, pulmonary edema, pleural effusion, and even congestive heart failure. Following fludrocortisone therapy, her serum Na concentration